ROLE OF ESCHAROCLYSIS IN MANAGEMENT OF DEEP BURN

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Year 2000

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CONTENTS

CHAPTER	PAGE NO.
INTRODUCTION	01-07
REVIEW OF LITERATURE	08-24
MATERIAL AND METHODS	25-34
OBSERVATION	35-47
DISCUSSION	48-59
CONCLUSION	60-62
BIBLIGROPHY	63-72
SUMMARY	(In Separate cover)

INTRODUCTION

INTRODUCTION

The problem of burn is as ancient as the time when man attempted to use fire. Burns have caused great suffering to mankind physically, socially as well as economical and still remain a major health problem. More than 2 millions suffer thermal injury annually all over the world. According to "Central Bureau of Health Intelligence" number of deaths by fire in 1984 was 15,741, which had increased up to 18,943 in 1988.

Thermal burns are caused by application of heat to the body. The degree of resulting burn injury intensity and duration of heat depends on application and conductivity of tissue involved. Congested living condition and wearing loose garments cause burn from wood stove, kerosene stove, kerosene lamp and leaking gas cylinders. Burning dress can produce a temp of about 100°C. In addition thermal injury is frequently observed in patients who have been exposed to direct contact with hot liquid, hot metal, toxic chemicals or high voltage electric current and explosion of natural gas, propane, gasoline and other inflammable liquids. In civilian practice, scald usually from hot water is the most common cause of burn. Water

at 140°F (60°C) creates a deep dermal or full thickness burn in 3 seconds. At 156°F (69°C) the same burn occurs in 1 second. Scald burns from grease or hot oil are usually deep dermal or full thickness burns. Cooking oil and gas may produce temp.of about 400°F. In scald burn, exposed area burns less deep than the burnt area covered with thin clothing. Clothing retains the heat and keeps the liquids in contact with the skin for longer period.

The cause and risk in burn injury as well as risk of burn death are influenced by age, economic circumstances, geographic location and season of year and occupation. Risk of burn injury and death is maximum in the very young, the elderly, the economically disadvantaged and in the winter months.

More than 90% of burns are caused by carelessness or ignorance and are completely preventable, about 74% are domestic burns and 79% of all domestic burns involve women and children.

The chemical burn, in the civil population is most commonly seen in industrial mishaps, laboratory accidents, civilian assaults and inexpert

application of agents used for medical purposes. The principle difference between thermal and chemical injury is the length of time during which tissue destruction continues. The chemical agent causes progressive damage even after inactivation, while thermal injury ceases after removal of heat source.

Ever since man burnt himself, he has covered the raw wound with a variety of medicaments in an effort to heal. Coverage of this raw expeditiously, still remains an inseparable part of treatment. Various mode of local treatment have been suggested and used from time to time but the dilemma continues and the search for an ideal agent continues. In ancient times the emphasis treatment of burn wounds was local application of various medicinal products like resins and bitumen vinegar, extracts of plants, honey and bran, gum, goat hairs and other funny things like milk from a mother who has given birth to a male child. Subsequently these local applicants changed to specific chemicals as tannic acid, silver nitrate, gention violet and petroleum gauze, but with the advent of anti-microbials like sulfamylon, sulphadizine cream etc. the emphasis turned to their local application.

The three basic concepts regarding the local problem of burn injury are: -

- 1. To protect microorganism invasion from without.
- 2. Burn injury provides a large raw area, which causes a loss of large amount of water, electrolytes and plasma proteins.
- 3. The problem of pain caused by irritation of exposed nerve endings by clothing, dressing or even the mere contact of air.

To minimize the evaporative effect of raw surface produced by burn injury, various biological and synthetic covering materials have been used by various workers at different times. Different biological and synthetic covering are homografts, skin heterografts, skin collagen sheets, amniotic membrane, solid silicone polymembrane, cotton gauze fabrics, sprays, gels and laminates etc. But a perfect wound dressing is still a dream because covering materials although being good dressing materials have their limitations and disadvantages like subgraft suppuration, limited availability, high cost and potential risk of transmission of diseases like Hepatitis.

To decrease the introduction of infection from without there are a number of topical agents which are used but almost all of them don't fulfill the criteria of an ideal agent. An ideal topical agent should have a wide spectrum of action, and should be least toxic to the patient, i.e. with minimal chances of development of resistance against drug. It must have water vapour transmission rate which will allow the proper moisture balance in the healing wounds i.e. to prevent either hydration or desiccation of the healing tissue and it should penetrate eschar.

The management of burn wound sepsis is still a very challenging problem in terms of morbidity and mortality. The improvements in infusion therapy has led to a clear reduction in mortality due to shock but the problem of infection is the major cause of death in burn patients. The vast advancements in medical science and availability of various broad spectrum antibiotics, has lengthened survival time but still late deaths due to burns are invariably associated with infection. The avascular nature of burn tissue as a result of thrombosis of vessels limits the delivery of endogenic phagocytic cells and also decreases efficacy of systemically administered antibiotics leading to propagation of

infection. In addition to infection, maceration and pressure necrosis also favor microbial proliferation and impair circulation. The problem of the deep burn is slightly more complex even adequately administered antimicrobials do not reach the subescharal plane prolonged ischemia. due to The systemically administered antibiotic can only reach the subescharal plane by gradient diffusion from the wound periphery. So they are always inadequate in preventing colonization of bacteria and hence it is that patients with deep burns have more seen extensive sepsis.

This local source of virulent organisms in presence of lowered body resistance can alter the fragile balance between resistance and infection leading to frank septicaemia and death at any time (Liedberg, N.C.F., Reiss, E and Artz C.P. 1954). Once generalized burn wound sepsis has developed, the chance of survival is 10% or less (Mc Manus WF. Goodwin CW, Jr. Manson AD Jr et al, 1981).

Thus, it is necessary that treatment of local infection be given top priority and since systemic delivery of antibiotics is sub-optimal, more

reliance is to be put on local methods of control of infection.

With this view and also because daily dressing would not be needed hence pain would be minimized. Povidone Iodine (Polyvinyl pyrrolidone) and Neosporin powder would be used in superficial and deep burns In this study in addition to the above, multiple subescharal injections of povidone iodine in subescharal plane would be given to counter the infection in subescharal plane by directly increasing the level of subescharal antimicrobials and help in early escharolysis by opening up subescharal plane and decreasing bleeding on separation of eschar.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Burn is one of the oldest form of injury and a universal problem. Even the prehistoric suffered from burns from lighting, molten lava and steam of hot springs. The Epidemiology of burns has gradually changed with time, reflecting changing environment of man. Electrical, chemical radiation burns have been the price industrialization. Tn addition homicidal and suicidal burns have increased as a result of social development. Over the years there has progressive refinement in the treatment of burns. All research is being aimed at promoting tissue healing and reducing the chances of septicaemia. It is very important to know how this outlook towards treatment of burns has changed over the centuries.

Ancient methods

Ancient methods of treatment in ancient era was mainly local application which was highly different and often exotic. In the 5^{th} and the 6^{th} century BC, Egyptians treated burn by mixture of gum, goat's hair and milk of a lady who had given birth to a son. Chinese and Japanese were using tincture and extracts from tealeaves in around 480 BC. Romans

had tried other innovative methods like honey and bran, vinegar and wine. The mixture of resin and bitumen was used by Hippocrate. He also used warm vinegar soaked dressing to relieve pain. In ancient Rome, Ceicus suggested a mixture of honey and bran local application. Gallar suggested local application of vinegar and wine on burn surface. In the 9th century AD Paulus of Aegina used various emolient preparations. A mixture of white lead, oil of roses and wax had been used by Rhozer (580-920 AD). Ice cold water was used on burn surface by Arabian physicians. Amrose Pare (1517-1590) suggested the use of ointment in treatment of burn. Clowes (1591) used 5 different complex preparations in the treatment of burn. Vinegar and chalk was used locally by David Clegron (1792). Pressure dressing as a mode of treatment was suggested by Edward Kentish (1797) to relieve pain and to stop blister formation. Ice cold water acts as a good analgesic and prevents oedema formation was suggested by Sir. James Earle (1799) and used in the treatment of burns.

The later to present day: -Between 1833 to 1868 the dry method of dressing remained supreme. This form of therapy almost replaced the purposeless use of carosine oil and Linementon aquae Calcis (Syme 1834 in his principles of surgery).

But in 1871 Copland suggested exposure method was best method of the treatment of burn.

The discovery of antiseptic properties of carbolic acid by Joseph Lister heralded its use in the treatment of burns in 1898. Carbolic acid oil (2.5%), was applied to injury area and dressing removed every day. The disadvantage was local gangrene converting the partial thickness burn into full thickness burn. The local absorption of carbolic acid caused adverse effects like nausea, vomiting, muscle twitching, excitement, weakness, hypotension and perspiration.

The period following carbolic acid era, was an important period called saline wet dressing antiseptic era extending between 1885 to 1910 for treatment of burns. Since then the various topical agents and treatment methods have been used in burn sepsis which include wet dressing with sodium bicarbonate followed by application of solution of picric acid or boric acid as advocated by Oppenheimer (1906). Picric acid absorption from local injury site may cause tachycardia, nausea, vomiting, diarrhea, fever, renal failure and coma, was recognized by A. Maclennen (1903) and E.J. Elliot (1906). On other hand boric acid may cause

rashes with desquamation of skin, restlessness, confusion, weakness, hypothermia, hypotension, tachycardia and renal failure.

Wet dressing gave way to wax as a modality of treatment. Wax containing 250 mg % Betanapthol was applied at a temperature of 50 to 60° C. This was recommended topically between 1910 to 1926. The adverse effects were extensive with renal and hepatic damage and convulsion and even death.

Local application of tannic acid on burn surface was started by Edward Clark and Devitson in 1925 at Henry Ford hospital. Later on in 1944 Maclux in the same hospital described it as a hepatotoxic agent and attributed many deaths due to its toxicity. Gention violet as an escharolytic agent in burn surface was tried by (Aldriage, 1933). Allen and Koch (1942) suggested the use of Petroleum gauze locally with strict immobilisation as further advancement in the management of burn injury in IInd world war "Exposure Method" of treatment of burn wound must have actually been used for several centuries. The credit for reintroduction of exposure method as modality for treatment of burn goes to Wallace (1949) of Edinburgh and Pualki, Artzi and Blocker (1950) of

U.S.A. Later on other surgeons accepted the same method with a view that formation of crust acts as a physiological covering of burn wound, thus reducing the disadvantages of raw area. Exposure causes drying of the wound and inhibition of bacterial proliferation, besides, ultraviolet light also acts as a deterrent to bacterial growth. The use of topical agent further enhances the control of bacterial growth already achieved.

Inspite of various treatment modalities advocated during the last century, none of them could significantly reduce the incidence of burn morbidity and mortality.

The simultaneous progress in other fields like improvement in microbiological techniques, culture media etc. aided in identifying the organism responsible for death. The principal cause of death was septicaemia and organism responsible was staphylococci (Ludbug Reise and Artz 1953). The world war had heralded a new era in the treatment of infection with the development of antibiotics like penicillin and other associated compounds in controlling most of the gram positive and gram negative bacteria.

Because of systemic delivery of antibiotics at the burn wound sites is sub-optimal, topical agent should play an important role in the management of burn injury. A number of techniques with topical agents have been evolved in mid 1960, that have substantially decreased the incidence of burn bound sepsis. Each has its own advantage and disadvantages.

BIODRESSING

The main cause of morbidity and mortality in burn patients is toxiaemia due to absorption of toxins from injured surface of burn invaded by micro-organism, so the epoch making discovery in the treatment of burns was the advent of 'Biological dressing' for restoration of impaired barrier. If an ideal skin substitute could be discovered, it could bring about a drastic change in the outlook to the treatment of burns. could be excised in early in post-burn period and then covered with skin substitute. In about a week or two the patients could be discharged. Autologous healthy skin grown in tissue culture can be used to replace this.

In brief an ideal skin substitute should possess the following properties -

- 1. Should adhere rapidly and strongly with the underlying raw areas.
- 2. Should have water vapour characteristics like that of normal skin.
- 3. Should be elastic enough to stretch freely over joints.
- 4. Should be durable.
- 5. An intact bacterial barrier.
- 6. Should not be antigenic.
- 7. Should be easy to apply and remove.
- 8. Should be cheap.
- 9. Should be homeostatic.

The various materials that could be used as a skin substitutes are -

I. BIOLOGIC:

- (a) Human allograft (Homograft) :
- Living donor
- Cadaver donor fresh
- Cadaver donor frozen
- Amniotic membrane

(b) Xenograft (Heterograft)

 Living donor fresh, Frozen radiated or dried.

(c) Tissue derivatives:

- Collagen sheet fabric or sponges
- Bioplast fibrin.

II. SYNTHETIC:

- Solid silicon polymer membrane
- Other plastics
- Microporous materials

III. COMPOSITE MATERIALS :

- Surface membrane :- (Silicon, microporous, trydron).
- Adherant substrate :- Collagen, cotton gauze, synthetic polymer, sponge,

 Vetour, flecking or fabric.

LIMITATION OF BIOLOGICAL DRESSING :

Inspite of being the best dressing material for burn wound, they have certain disadvantage in form of -

- 1. Paucity in availability of autograft & other dressings.
- Overwhelming financial overtones (which turn out to be an important consideration in a poor country like India).

- 3. Subgraft suppuration.
- 4. Transmission of disease likes hepatitis.
- 5. Lyophilised allograft skin shows fewer adherence to the wound and is of sufficient thickness, undergoes dermal separation and causes desiccation of exposed dermis.

Problem of Burn Wound Infections and Topical Agents

The management of burn wound sepsis is still a very challenging problem in terms of morbidity and mortality. It is the major factor causing death, during the phase of illness. Because the systemic delivery of antibiotics at the burn wound site is sub-optimal, topical agent should play an important role in the management of burn wound sepsis. There are a number of topical agents used in burn sepsis. Yet the search for ideal topical therapy has as yet proved elusive. Over the years the endeavours aimed at topical therapy have been topical oint (Fox CL Jr, Rappole B.W and Stainford W 1969), early escharotomy or skin grafting (Burke JF, Bondoc CC and Quinby WC 1974) or amniotic membrane application (Bose B 1979).

The properties of ideal topical applicable drug, given by Zellner P.R and Buggi S. in 1985, is as follows -

- (1) The topical agent should have antiseptic properties and wide spectrum of action.
- (2) Minimal development of resistance against drug.
- (3) Must penetrate the eschar.
- (4) Must not harm the viable tissue.
- (5) Should not hinder the growth of proliferating tissue.
- (6) Must be nontoxic and must not interfere with metabolism.
- (7) Must not be antigenic.
- (8) Should be cheap.
- (9) Must have tanning effect.
- (10) Should be easy to administer and to remove.

To asses the effectiveness of topical agents following parameters are used:-

- Surface culture and sensitivity reflect bacterial infection on the surface of burn wound.
- 2.) Quantitative estimation of bacterial counts indicating the degree of infection.
- 3.) Culture of burn wound biopsy provides three parameters for assessment of infection.
 - A.) Quantitative bacteriology.
 - B.) Qualitative bacteriology.

C.) Histology:-They provide perivascular, perilymphatic and intraluminal accumulation of bacteria prove without doubt invasive bacterial infection (Krupp S, Baechler M, Bille J 19850).

In order to fulfil the above criteria, a number of newer techniques of burn dressings have been evolved. Each has it's own advantage and disadvantages. A few important agents, some of which are still in vogue like Mefamide or Sulfamylon (Moncrief 1974), cerium nitrate (Williams W Monafo 1975), 0.5% silver nitrate (Mayer 1960), silver sulphadiazine (Fox CL Jr 1975) and Mercurochrome. These topical agents have more of palliative effect and help only in reducing the local bacterial concentration (from 107 to104 microbes per gm.), not eradicating the bacteria from the wound (Artz C.P 1979). These agents have deleterious side effects, and can even prove lethal, if the patient is not carefully monitored, for example silver nitrate causes necrobiosis, discolouration of local area due to precipitation of silver ion, serum Na+, K+, and Cl- deficits and alkalosis (Kulick al 1980). Hence the monitoring the Na⁺ , K⁺, Cl⁻ becomes a must. Sulfamylon, although readily diffuses through the eschar, is

potent inhibitor of carbonic anhydrase and it may induce acid base derangement leading to acidosis. With Mercurochrome, toxic amounts of mercury can be absorbed (Steen 1983).

Due to side effects of silver nitrate, another topical anti-microbial Silver Sulphadiazine (S.S.D) introduced. S.S.D exerts an anti-bacterial action mainly against Pseudomonas, poorly penetrates the eschar, the eschar does not adhere to the dressing and the silver ions are released slowly in a concentration such that they are selectively toxic to pathogens (Rosenksaz 1972). Silver ions act on bacterial cell surface causing alteration in cell wall and cell membrane leading to death. The disadvantages are crystalluria, resistance to sulphonamides and high cost adverse reactions like burning, rashes, itching and difficulty in daily application on burn surface. But overall SSD is distinctly superior to silver nitrate

Povidone-Iodine (PVP) has been in use for over a period of four decades. (Garner et al in 1959, Georgide in 1962, Connell in 1964, Copeland in 1972). Nicholas G. Goergide in 1972 found PVP to be one of the best antiseptic agents because of its broad spectrum, good penetrability through the eschar while exerting its microbicidal effect. It

does not harm the surviving and proliferating tissue and has neither antigenicity nor any skin reaction and infecting microorganisms do not develop resistance to it. It also has tanning effect and enhances wound healing by preventing infection.

Povidone-Iodine can easily be applied locally or along with Aserbine (Knock, D.M 1985). P.V.P is effective against a wide range of gram positive and gram negative organism as well as some fungi, spores and viruses (Schwarts, Shires and Spencer, 1988). Prolonged treatment with povidone-Iodine does not have any effect on thyroid gland. But in 1974 Law EJ and McMillan reported adverse effect of PVP on thyroid gland in two patients out of seventy patients.

In 1985 Balogh D, Bauer M and Riccabona G reported that patients treated with PVP may show increased serum iodine level but simultaneous measurement of thyroid hormones failed to reveal any significant impairment of thyroid function apart from depressed T₃ coupled with increased reverse T₃. They also reported that if renal function is unimpaired, the absorbed iodine is quickly excreted and no clinical sign of iodine toxication were observed. In 1991, Nakano S, Uchiyam A of Japan reported that prolonged exposure

to wet PVP can cause chemical burn but no other author observed such type of reaction in their study.

Iodine released from Idophore (Polyvinyl Pyrrolidone) after application of PVP, precipitates the proteins of bacteria and other micro organisms and reacts with exudates to precipitate proteins on wound surface forming a firm crust under which no micro organism can survive.

The efficacy of PVP increases when used with Neosporin powder. Neosporin powder (Wellcome & Burrough's) contains three ingredients:-

- 1.) Neomycin sulphate.
- 2.) Zinc Bacitracin.
- 3.) Polymyxin-B.

Neomycin is predominantly a locally acting bacteriocidal drug, having adverse effects like otonephrotoxity and depression of respiratory system. Bacitracin is effective against grampositive cocci and bacilli and Polymyxin-B is antibacterial against mainly H. pertusis, Pseudomonas, E.coli and other gram negative bacilli and has side effect like acute renal failure and nystagmus. Its systematic absorption is little and allergic reactions are rare.

Because PVP + N was applied as a "crust", because the crusting effect ensures that the locally applied drugs remain at the burn site for a prolonged period and at the same time because subsequent application are applied over the previous one, the patients does not feel any pain first application. Even the first the application entails only minimal pain. In our opinion, if burn patients can be given an option of pain free local burn own management it would be of immense psychological relief to the patient. addition, nursing personnel time is reduced by about 70 to 80% on average and therefore treatment cost is less.

In qualitative bacteriology immediate gram staining shows the presence of bacteria and identification of bacteria from wound biopsies provides precise identification. The organism which have been recovered from culture of wound swab and wound biopsies are as follows:-

- A.) Gram negative bacilli Enterobactor spp., Klebsiella, E-coli, pseudomonas aeroginosa etc.
- B.) Gram positive bacilli Enterococci, staph, epidermis, staph. aureus, streptococcus.

C.) Other opportunistic infections - Fungal eg. Candida spp., yeast and viral infection.

The combination of drug treatment utilizing the PVP + Neosporin powder may act as complimentary to each other. Beneficial encouraging results were seen with this combination in term of infection, role & markedly reduce healing time of burn wounds by Sinha R et al in 1988. Following study using multiple subescharal injection of PVP was done.

The subescharal technique is based on original observation of Drs & Moncrief demonstrating the ischemia with in a burn wound and the failure of nutrients or systemic and local treatment to reach the subescharal plane. The systemically administered antibiotics can only reach the ischemic area by gradient diffusion from the wound periphery.

Baxter et al 1973 first used subescharal injections of antibiotics in deep burn. Subescharalclysis offers the theoretical advantage of direct administration of antimicrobial to deep burn area where systemic antimicrobials fail to reach. Subescharal injections of PVP helps to decrease the ultimate bacterial concentration in the subescharal plane and help in early escharoclysis, decreasing the bleeding on

separation of eschar and ultimately in preventing septicaemia and death.

In the present study our aim is to assess the efficacy of subescharal injection of P.V.P with surface P.V.P + Neosporin powder, application in term of

- Subescharal bacterial concentration,
- Escharolysis time.

At the same time to see the effect of P.V.P on thyroid function/renal function.

MATERIALS AND METHODS

MATERIAL AND METHODS

The present study was conducted at M.L.B Medical College, Jhansi from April 1998 to Feb 2000. In this study we included patients with deep burns with or without partial thickness burn. All patients of the burn were divided in to two subgroups matched by age and percentage of burn. All patients were treated topically with topical povidone-Iodine lotion (5% w/v) and neosporin powder. In the test group we additionally injected PVP solution by diluting it in equal volume of normal saline (i.e.0.25% available iodine solution) in subescharal plane.

MATERIAL:

Povidone Iodine lotion:

- (1.) PVP lotion with 0.5% available iodine for topical application in equal amount.
- (2.) PVP lotion diluted in saline 0.25% available iodine for subescharal injection.

Neosporin powder : (Wellcome and Burrough) -

Neosporin powder is available in powder form, 10 gr. pack are available in the market. This powder contains the following three ingredients.

- 1. Polymyxin B sulphate Each gram of neosporin powder contains 500 U.B.P. of polymyxin and sulphate.
- 2. Zinc Bacitracin Each gram of neosporin powder contains 400 U.B.P. of zinc bacitracin.
- 3. Neomycin Sulphate Each gram of neomycin powder contains 3400 U.B.P. of neomycin sulphate salt.

Neosporin powder was used for sprinkling over the burn area till a uniform coating of powder was obtained.

Selection of patients

All patients of deep burn of up to 50% body surface with or without partial thickness burns who came to emergency ward or O.P.D of this hospital were included in this study irrespective of their age, sex, socio - economic status, contamination of wound and mode of injury.

Method of Study

The selected cases were subjected to detailed history and physical examination which were recorded on following lines:

1. History:

- Introduction, name, age, sex, occupation, rural/ urban, address, date of admission, date of discharge.
- ❖ Regarding the burn accident date and time of burn, hospital attendance delay time, place of accident, cause of burn and treatment given prior to admission (if any).

2. Physical Examination:

❖ General Examination : The patients were examined for general condition, pulse rate, blood pressure, temperature, respiration and hydration.

❖ Local examination :

- A. Percentage of Burn: It was calculated by Wallace Rule of Nine in the adult and "Lund and Bowder Chart" in children.
- B. Depth of Burn : Superficial/ deep.

Estimation of Depth of burn :

The hypodermic needle was used to see for the pain sensation. The area with increased sensibility was considered to the partial thickness burn. The area with marked reduced or absent pain sensitivity was considered to be deep or full thickness burn. This was also confirmed by pulling out a hair from burn surface. On the 3rd degree or deep burn, hair pulls out easily and painlessly. The latter test is of value in borderline cases of second degree form. In addition help of the following criteria was also sought.

Classification of Burn	Appearance of Burn Area	Pain Sensation
1 st degree	Erythematous	Painful and
		hyperaesthetic
2 nd degree – A	Blister with reddened base	Painful and
		hyperaesthetic
В	Blister with blanched base	Painful hyperaesthetic
		(or) anaesthetic at place
3 rd degree	Leathery pale or pearly	Painless and anaesthetic
	white or charred dry	

The 1^{st} and 2^{nd} - A were graded as superficial and 2^{nd} - B and III were considered as deep burn.

Estimation of Percentage of Burn :

Wallace rule of Nine

The percentage of burn depicted against each area in adult.

Area	Anterior %	Posterior %	Total %
Head & Neck	4.5	4.5	9.0
Upper limb (Single)	4.5	4.5	9.0
Trunk	18.0	18.0	36.0
Lower limb (Single)	9.0	9.0	18.0
Genitalia			1.0

Lund Browder Chart:

The percentage of burn depicted against each area in different age group in children.

Area	1 year	5 to 9 year
Head	19.0	13.0
Neck	2.0	2.0
Anterior trunk	13.0	13.0
Posterior trunk	13.0	13.0
Buttock	2.5	2.5
Genitalia	1.0	1.0
Arm	4.0	4.0
Fore arm	3.0	3.0
Hand	2.5	2.5
Thigh	5.5	8.0
Leg	5.0	5.5
Foot	1.5	3.5

Contamination of Wound :

Apparently clean : No contamination of

foreign body, clean

intact, blisters.

Mild contamination :Slight contamination,

ruptured blisters, open

wounds.

• Gross contamination : Heavy contamination with

dirty cloth, foreign

body, pus, dust and/or non

medical substance i.e.

cow dung etc.

Resuscitation and general treatment: Patients were resuscitated by maintaining airway, I.V. fluid supplementation, plasma and blood infusion, analgesic, antibiotics and tetanus prophylaxis according to the need of patients.

LOCAL MANAGEMENT OF WOUND :

Preparation of burn surface: A swab from burn surface was taken for culture and sensitivity test. Patient was given necessary sedation. A gently but thorough debridement of wound done by removing necrosed skin and blisters. The area was tested for

degree of burn. Then the whole burn surface was cleaned with sterile normal saline thoroughly.

Application of PVP & Neosporin powder on burn surface:

In all patients the application was started by cleaning with saline, then sprinkling a uniform layer of neosporin powder on burn surface. Over this the solution of Povidone Iodine (0.5% available iodine) was sprayed uniformly. This application help in forming crust on superficial burn area. On the first day, three such applications were carried out without removing previously applied layers. On the second day, the application was reduced to two and from the third day onwards, this application was limited to those area from which the crust was either separated or cracked. Subsequently those areas showing discharge with infection were subjected to twice daily applications, each time after removal of infected crust.

Method of subescharal injection PVP lotion in test group:

In the test group patients with deep burn, we followed the same procedure as mentioned above. In

addition we injected PVP solution by diluting it with equal volume of normal saline and injected at multiple sites in the subescharal plane as 0.25% solution w/v by hypodermic needle. Each injection site would receive one ml this solution and cover one square inch of burn surface. The injected amount was 13 ml in 1% deep burn in adult. This injection was started on 7th post burn day and repeated twice weekly until escharolysis was completed.

Follow up of patients.

The assessment of result was done by utilizing subjective and objective parameters with patients examination visits and investigations.

Subjective parameters.

The patients was asked about:-

- 1.) Pain and discomfort (mild, moderate, severe).
- 2.) Fever and palpitation.
- 3.) Any evidence of allergy as itching, rashes, nausea and vomiting.

Objective parameters:

Observation for following was done:-

- 1.) Appearance & duration of deep burn area.
- 2.) Presence of discharge or soakage.
- 3.) Surface colonization of bacteria qualitative analysis.
- 4.) Subescharal colonization of bacteria qualitative and quantitative both.
- 5.) Eschar separation time.
- 6.) Wound status for grafting.
- 7.) Graft acceptance.
- 8.) Thyroid status-serum T3, T4, TSH levels.
- 9.) Renal function-serum creatinine and blood urea.

WORKING PROFORMA

	Case N	O	•••	
Name:		MRD No		• • •
Age/Sex:		Ward/bed		
Address:				
Date of admission Hospital attendance delay time Mode of burn	: :			
INVESTIGATION:		At the time of admission	7 th day	18 th day
Surface culture	•			
Subescharal culture	•			
Escharal biopsy				
Γ3, T4, TSH levels	:			
Serum creatinine/Blood urea	•			
Eschar separated on	**************************************			
Total amount of PVP 0.25%/% burn				
Total amount of PVP+Neosporin Powder/% burn surface area				
Graft applied on				
Discharged or other				

OBSERVATION

The present study comprised of 118 patients of deep burns with or without superficial burn admitted in indoor and emergency ward of M.L.B Medical College, Jhansi. These patients belonged to different social strata and were of both sexes and age varied from 1 to 50 years.

All patients included in this study had burn involving 50% or less than 50% body surface area. More than 50% of deep burn and more than 70% of mixed burns were excluded from the study because of high mortality.

Table -1
Showing sex incidence in burn cases:

Sex	Number of cases	Percentage	
Male	40	33.89	
Female	78	66.11	
Total	118	100.00	

Table 1. Shows that out of total 118 cases studied there were 40 males (33.89%) & 78 (66.11%) female with M:F ratio of 1 : 2 approx..

Table -2
Showing mode of burn:

Mode of Burn	Number of cases	Percentage
Thermal Burn	112	94.92
Electrical Burn	6	5.08
Chemical Burn	•	-
Total	118	100.00

Table 2. Shows that most of the patients had thermal burn (94.92%) during cooking or leakage of gas or bursting of stove etc. while only6 patients (5.08%) had electric burn. No patient suffered from chemical burn.

Table - 3
Showing the age incidence in burn case.

Age group (years)	No. of cases	Percentage
0-10	5	4.23
11-20	23	19.49
21-30	52	44.06
31-40	24	22.33
41-50	10	8.47
>50	4	3.38
Total	118	100.00

Table 3. Shows that maximum (99 cases out of 118, 83.89%) were in the range of 11 to 40 years. Only 5 (4.23%) of total were below 10 years of age while only 14 (11.86%) were above the age of 40 years. So we can say that persons in their active years of life are much prone to sustain injury as compared to childhood and old age.

Table - 4

Age & Sex incidence

Age group (years)	Male	Female	Total
0-10	3	2	5
11-20	8	14	23
21-30	14	38	52
31-40	9	16	24
41-50	4	6	10
>50	2	2	4
Total	40	78	118

Table 4. Also shows that in the age group of 11 to 40 years females suffered from injury two times more than male of same age group.

Distribution of cases according to location of burn accident & sex

Table - 5

Location						
of burn	Male		Female		Total	
accident	Number	%	Number	%	Number	%
Indoor	12	30.00	74	94.87	86	72.88
Outdoor	28	70.00	4	5.13	32	27.12
Total	40	100.00	78	100.00	118	100.00

Table 5. Shows that most of burn accident occurred in indoor location as a whole 72.88% (86 cases out of 118). Among female cases 94.87% cases sustained burn injury at home.

Table - 6

Distribution of cases according rural & urban incidence in burn case.

Rural/Urban area	Number of cases	Percentage
Rural	86	72.88
Urban	32	27.12
Total	118	100.00

Table 6. Shows that incidence of burn injury was more than two times (86) more in rural population as compared to urban (32) population.

Distribution of cases according to duration from injury to admission to hospital

Table - 7

Duration	Number of Cases	Percentage
0-1	68	57.62
1-2	24	22.34
2-5	13	11.01
5-7	7	5.93
>7	6	5.08

It is clear from table 7. that out of total 118 cases, 57.62% reached to hospital with in 24 hours of burn accident. Only 6 cases (5.08%) reached the hospital after 7 days of accident.

Table - 8
Treatment protocol

% of deep burn	Test group	Control group	Total
< 15	6	4	10
16-25	20	8	28
26-35	38	16	54
>35	16	10	26
Total	80	38	118

Table 8. Shows that all patients were divided into two subgroups matched by age and percentage of deep burn. Test group consisted of 80 (67.79%) and control 38 (32.20%) patients. Most patient presented with 16 to 35% of deep burn. Patients of control group were treated with PVP solution + Neosporin powder. 80 patients of test groups were treated with same procedure with addition of subescharal injection of PVP at multiple sites.

Table - 9
Surface culture report.

Organism	Test group			C	Control group		
	1*	2**	3***	1*	2**	3***	
Staphylococcus	38	22	8	21	14	9	
Streptococcus	10	6	01	08	03	02	
E.coli	24	13	06	12	08	04	
Klebsiella	8	3	01	6	02		
Proteus	16	7	02	9	04	01	
Pseudomonas	20	5	02	7	02	01	

^{*} At the time of admission.

^{** 7&}lt;sup>th</sup> day of post burn.

^{***} 18^{th} day of post burn .

Table - 10
Eschar culture report.

Organism	Test group		Control group	
	7 th day	18 th day	7 th day	18 th day
Staphylococcus	22	08	14	08
Streptococcus	04	01	02	01
E.coli	13	06	09	06
Klebsiella	01	•	02	•
Proteus	02	01	02	01
Pseudomonas	02	01	01	01

Table 9. & 10. Shows that, in all patients surface culture were taken for qualitative analysis at the time of admission, $7^{\rm th}$ day and on $18^{\rm th}$ day and escharal culture and biopsy were taken on $7^{\rm th}$ and $18^{\rm th}$ day. The most commonly involved organism were staphylococcus, E.coli, streptococcus.

Table -11

Bacterial count after treatment.

Bacterial	Test g	group	Control group		
Count	7 th day	18 th day	7 th day	18 th day	
$<10^{5}/gm$	52	72	15	22	
>10 ⁵ /gm	24	04	21	13	

Quantitative bacterial analysis showed that bacterial count below $10^5/\mathrm{gm}$ was markedly better in PVP + N + Subescharal injection patients at 7^{th} days post treatment. On 18^{th} days significantly more patients showed bacterial counts below $10^5/\mathrm{gm}$, compared to the control group.

Table - 12

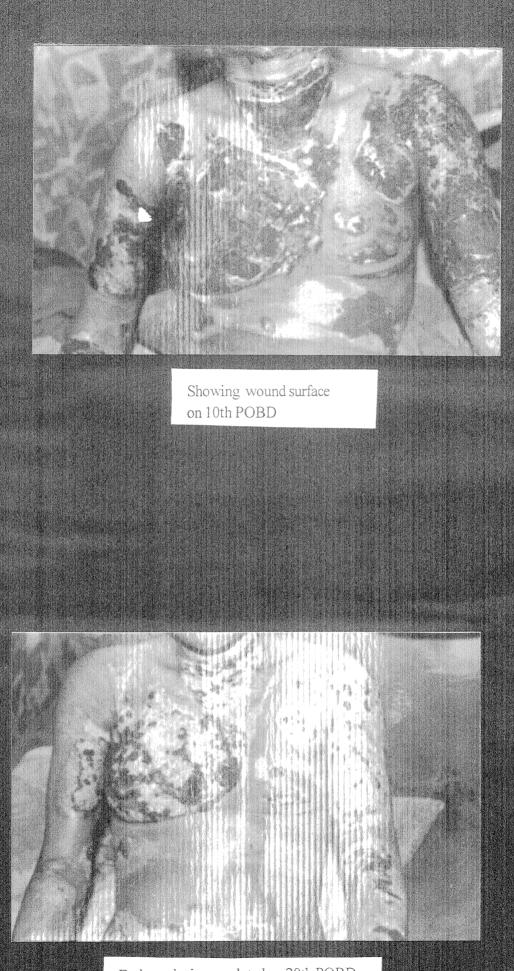
Time taken for escharolysis.

Percentage	Time in days					
of	<20	21-30	31-40	>40		
deep burn						
Test group						
<15%	6	-		_		
16-25%	18	1	_	-		
26-35%	27	8	-	_		
>35%		5	3	<u>-</u>		
Control group						
<15%	02	4	-			
16-25%	01	7	02	-		
26-35%		6	8	<u>-</u>		
	<u>-</u>	<u></u>		02		
>35%						

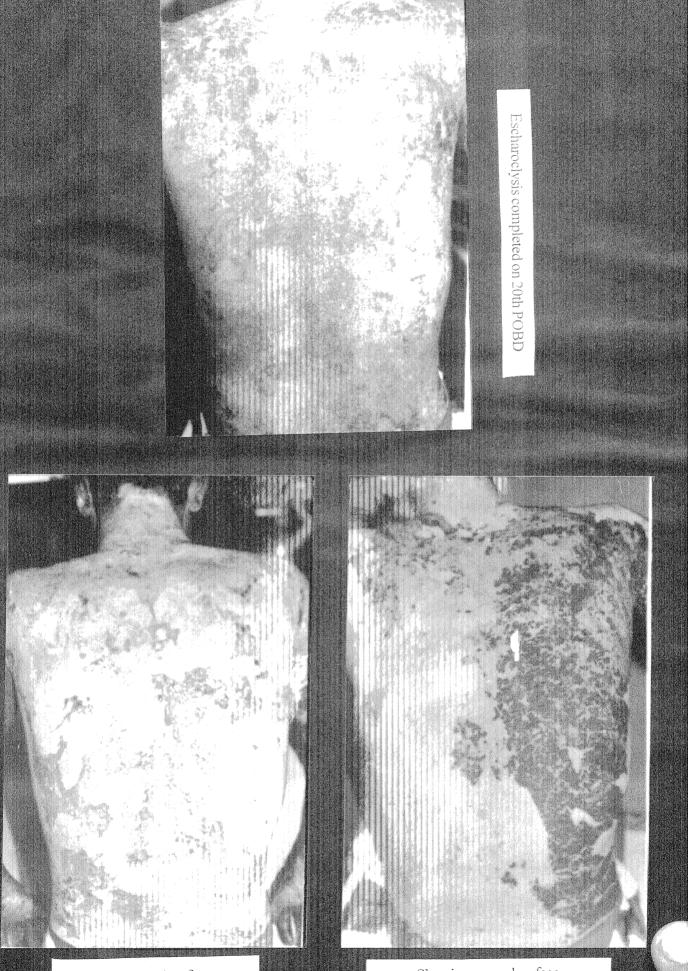
Table 12. Rate of escharolysis most of patients when treated with PVP + Neosporin + Subescharal multiple injection showed complete escharolysis within 20 days, while patients treated only with PVP + Neosporin showed delayed escharolysis more than 30 days. In both groups with increase in the percentage of burns the time for escharolysis also increase.

Again from above tables it's clear that the time taken for escharoclysis was much less in the test group as compared to the control group.

Hence the treatment of deep burn with PVP + N + subescharal injection is distinctly superior to the topical treatment with PVP + N alone.









Showing wound surface on 7th POBD



Showing wound surface on 15th POBD



DISCUSSION

Burns are notorious in the sense that they break the continuity of skin and produce raw area, which is prone for invasion by micro-organisms and abnormal loss of body constituents. The water retention ability of skin depends on its effective vapour pressure and diffusion barrier offered by keratin layers and lipid contents in the stratum corneum. This lipid is thermolabile, when this barrier is removed after thermal injuries, the effective vapour pressure gradient is increased by 15-20 times. This results in a large amount of evaporative water loss amounting to an increase of 3-10 times the normal rate in sensible water loss (40ml/hr), the amount and duration for which the loss persists depends on the depth and percentage of burn.

In the treatment of burn injury the, the focal points are control of shock and infection and skin grafting (Peter Zellner). The improvements in infusion therapy in burn patients have lead to clear reduction in mortality (Zellner & Metzger 1976). There is consensus that infection is the primary source of morbidity and mortality from extensive burn injury in the present era. Bacterial infection has always been considered one of the most serious complication of burn injury.

Burn sites are potential areas for entry of hetrogeneous microflora with which human co-exist. The problem is further compounded by the severe depression of host immunological response to a degree directly proportional to the severity of burn.

The avascular nature of burn tissue as a result of thrombosis of vessels, limits the delivery of endogenous phagocystic cells and systemically administered antibiotics may not always achieve an optimal burn wounds\ concentration and, thus, may not be able to limit the injection of that site. Early closure of burn wound. Logically would limit the site of entry of infection but this is not always achievable. In addition to the infection, wound maceration and pressure necrosis also favour microbial proliferation and impairs circulation. At deep burn sites due to prolonged ischemia in the sub-escharal plane, systemically administered antibiotics reach only by diffusion gradient from the wound periphery (Koch DM 1985, William WM, Bruce F 1987, Yurf RW, McManus AT et al 1984). The large raw area produced by the burn wound with it's exudate or serum, works like a huge culture plate on which organisms can multiply uninhibitedly. The superficial burn can convert to a deep burn in the presence of infection. Thus the burn wound despite

the use of topical antibacterial agents remains a constant, potential source of systemic sepsis until eschar separation is complete, (Charles R. Baxter et al 1973). Once generalised burn wound sepsis has developed, the chance of survival is 10% or less (Mc Marus WF et al 1981).

Although it is still thought that burn wound can be virtually sterile at the time of injury, this concept is purely academic. Adequate sampling of burn surface will reveal bacteria in every instance, although during the first few hours the concentration may be very low (Robert B Lindberg).

In large burn areas, dense colonisation of pathogens can occur within 24 hours (William WM, Bruce F 1987 and Krupps et al 1985). In untreated patients, immediately after injury few bacteria can be recovered and these are predominately gram positive. The type and density of organisms present in the untreated burn wound change with time. By the fifth post burn day, pseudomonas can be recovered (William WM, Bruce F 1987). By the middle of the second post burn week the burn wound organisms are predominately gram negative, rods and fungi especially candida (Artz Cp et al 1979, Order & Moncrief 1965, William W Monafo, Bruice F 1987). The organism penetrates the eschar by migration and extends down to the viable-nonviable tissue

interface. At this site further microbial proliferation commonly occurs and promotes lysis of the denatured collagen and spontaneous sloughing of the eschar. (Order & Moncrief 1968). In patients with inadequate host defence capacity or those in whom the topical therapy is ineffective, the subescharal organisms invade the underlying unburnt tissue and may spread systemically (Moncrief AJ, Teplitz C 1964, William WM, Bruce F 1987).

Evaluation and treatment of the microflora in the burn wound to prevent septic complication are a challenging clinical problem. Bacterial colonisation of the burn wound may reach a concentration of 106 to 107 per gram of burn tissue before changes are evident either in the appearance of the burn wound or in the detection of clinical signs or symptoms of systemic sepsis (Charles R Baxter at al 1973). Surface culture techniques fail to predict accurately the presence or progression of burn wound sepsis due to poor correlation between the surface colonisation of the eschar and subcutaneous tissue (Bretano L & Gravens AC 1967, Clarkson JG, Ward CG & Polk HC 1967, Colebrook L, Lowbary EJL and Hrust L 1960, Georgiade NG et al 19669). Multiple eschar biopsy obtained serially from representative area of burn wound and culture

quantitatively and qualitatively furnish valuable information about bacterial growth. The literature recommends site a bacterial count or 105/qm of tissue as the upper limit for minimal penetration to deeper tissue level (Moncrief AJ, Teplitz C 1964, Krupps, Barchler M, Bille J 1985). Ιf concentration of the bacteria is >105 bacteria/gram of tissue, burn wound sepsis is generally present. Under such conditions skin grafts are often autolysed by infection. Therefore, prior to skin grafting, aggressive wound treatment must be instituted and continued until the bacterial concentration of wound biopsies falls <105 bacteria/gram tissue (Artz, Moncrief & Pruitt 1979, Parks, Linares & Thomson 1981, Robson, Krizek & Heggers 1975, Steen 1983, Teplitz 1969, Teplitz 1974).

Considering all the above facts we used PVP+ Neosporin powder topically in all patients. This combination forms an almost complete barrier against microbials (Sinha et al 1987). Neosporin powder contains polymyxin, neomycin and bacitracin. The combination of povidone iodine and neosporin was basically selected for both its physical and bacteriological properties. Povidone iodine has wide antibacterial, anti-fungal, sporicidal and viricidal properties (Zellner and Bugyi 1985, Robson CM,

Schaerf RHM, Krizep TJ 1974, Law EJ, McMillan BC 1972, William WM, Bruce F 1987, Georgiade NG, Harris WA 1973, Peter Zellner 1985). Neomycin bacitracin supplement the gram positive antibacterial properties and polymyxin protects against P.aeruginosa (Georgiade NS, Harris WA 1973) but not against staphylococcus aureus and haemolytic streptococci (Jackson DM, Lowbury EJZ, Topley E 1951). The tanning effect of PVP has an added advantage on dead layers of skin, creating a demarcation between viable and nonviable areas. The tanned second or third degree burns were never transformed into sticky necroses. In addition, the tanned skin is less likely to produce infected material that can be transported into lymphatic and blood vessels during surgical scraping (Peter Zellner 1976), so by this effect PVP keeps the surface dry holding colonization to low levels and also permits early surgery (Zellner PR, Bugyl S 1985).

Daily spray of PVP lotion and neosporin powder on the wound forms a "crust" which sets up a barrier to colonization of bacteria, helps in healing with limitation of infection. Because the 'crusting' effect ensures that the locally applied drug remains at the burn site for a prolonged period and at the same time because subsequent application are applied

over the previous ones, patient does not feel any pain after the first application. Even the first application entails only minimal pain. In our opinion, if a burn patient can be given an option of pain free local burn wound management, it would be of immense psychological relief to the patient. In addition, nursing personnel time is reduced by about 70 to 80% on average and therefore treatment cost is less.

By the above discussion, the presence of infection should be a consideration in deciding on the optimal time for surgery. If there is no or minimal infection, we can postpone the surgery and can grant time for removal of eschar in their normal course instead of doing surgical esacherectomy and can prevent haemorrhage. Deep burns start to lose their eschar in 2 to 4 weeks. In the presence of infection, eschar separation is early because of collagenase production from bacteria, but because of poor presence of granulation tissue and >105 bacteria/gram of tissue skin graft can not be applied to covered the wound.

Charles R Baxter et al in 1973 first used antibacterial therapy in the escharal plane by subescharoclysis. This method permits the delivery of high concentration of specific antibiotics into

the avascular burn wound interface by multiple subescharal injection. Later William F McManus et al (1982) also used antibiotics in the subescharal plane. But one thing was common in all studies that antibiotics solution was used in the presence of infection i.e. bacterial count >105 gm i.e. when the patient was critically ill none of them used them prophylactively or in the earlier course of wound infection. Sinha et al in 1988 in his study used injection of PVP subescharally first time in all patients of deep burn. That time also a controversy was there as to whether such a procedure (routine PVP-I subescharal injection) was helpful or not? Subescharal PVP injections were attempted basically PVP has been shown to have beneficial because antibacterial effects when used simultaneously, intrapleurally or intraperitonealy without any serious iodine toxicity (Zomoral J 1984). The concentration of .25% PVP may seem to be too low for it to be effective but it has been mentioned that with this concentration there is an increase in free iodine and antibacterial activity (Zomora LJ 1984). In our study we found that patients treated with multiple subescharal injections of PVP showed remarkably reduced subescharal bacterial colony count as compared to the control group. That subescharal injection of PVP was beneficial was

evident from the result; only one septicaemic mortality occurred in the test group of patients. The second beneficial effect is that it opens up a subescharal plane by tanning of non-viable tissue & reduced sticky necrosis thus helping in early esharoclysis and decreasing bleeding. Additionally tanned skin produced least infected material so that transport in lymphatic and blood vessels minimum. The burn wound in most of the patients were grafted immediately after the eschar separation because most of the patients showed a colony count far less than 10^5 /gm which is the upper limit below which grafting can easily be done without much fear of graft rejection. In the control group a considerable period of time was spent in limiting the infection at the burn site and infection and less granulation tissue was the cause of graft rejection.

However, reservations have been expressed by some authors that the large quantity of PVP-I absorbed during treatment can have an effect on the thyroid gland in non-euthyroid patients and can cause thyrotoxicosis and thyroid dysfunction. Law EJ and McMillan BG 1974 reported thyroid toxicity in 2 patients after using PVP lotion (1-% available iodine). The high serum iodine concentration can also trigger renal insufficiency but Zellner PR and

Bugyi S in 1985 in the study of approx. 1500 patients never saw thyroid & renal dysfunction in patients treated with PVP lotion, who were examined several time over a long period of time. In our study non of the patients showed any clinical evidence of iodine toxicity.

Zellner PR and Bugyi S in their study with PVP-I in burn patients, measured T3, T4, TSH and the iodine level in blood and urine over a period of 21 days. The result showed an initial steep increase in the iodine level of blood and a parallel rise in urine. This increase reached their maximum on second or third day. The high level of iodine is an indicator of the good penetration of PVP-I through the burn wound. Thereafter, the serum iodine concentration falls and a little later the urine concentration level falls as well, despite continued administration. An explanation for this phenomenon was given that it could be that either the application of the ointment was less or that the healing process was preventing penetration. Most probably penetration was less because of the tanning effect. The iodine levels returned to normal about one week after withdrawal of treatment. Initial decrease in T_3 and T_4 pointed to transient hypothyroidism due to inhibition of deposition because of high serum iodine

concentration. However, a gradual decrease in T_3 and T_4 was also observed in burn patients by Becker et al 1980 who had never received topical treatment with iodine so one can say that this change can be brought about by different kinds of stress or disease (Burger et al 1976). Furthermore, the thyroid hormones bound to proteins also become lost in large quantities because of the high protein loss in burn patients due to exudate or oedema.

They have also shown the absence any thyroid or renal toxicity with PVP application. Even the component of neosporin, especially polymyxin and neomycin, which all have serious systemic toxicity, did not show any toxicity because they are applied locally and there is limited absorption, preventing systemic toxicity. In addition, the combination of PVP + N also reduce costs and improve patient compliance, which to our mind are very important aspects of the management of burn patients.

Sometimes PVP-I injection causes slight pain to patient when we injected either in wrong plane or too much amount is injected or the eschar is attached too tightly.

Hence it can safely be concluded that subescharal injection of PVP is effective in decreasing bacterial count in subescharal plane,

early scar separation and adding possibility of early grafting and it's better acceptance.

CONCLUSION

The comparative effects of subescharal multiple injections of povidone iodine with surface PVP + Neosporin application were studied and compared in 118 patients. Out of which 80 patients were included in the test group and 38 patients in the control group. This study was conducted in-patients of deep burn with or without superficial burn or less than 50% of body area involvement who came to M.L.B. Medical College, Hospital, Jhansi. Conclusion drawn from the present study are as follows:-

- 1. Females predominant in the age group of 11 to 40 years of age.
- 2. Most of burns are thermal in nature.
- 3. Most of the burn accidents took place in indoor activity.
- 4. Maximum burn injuries occurred in rural area.
- 5. Patients of urban area mostly belonged to middle class families.
- 6. Patients with major burn reach directly to Medical College Hospital, and much earlier then those with minor burns.
- 7. No allergic reaction was observed in topical application or injection of povidone iodine.

- 8. Deep burn involving smaller area healed without a scar.
- 9. Scar were yellowish tinged hyperpigmented and fibrous in PVP + Neosporin treated patients.
- 10. Contracture was observed in 4 cases of test group and 2 cases of control group.
- 11. The serum creatinine, blood urea and T_3 (triidothyronine), T_4 (thyroxine) and TSH (thyroid stimulating hormone) values remained within in the normal range in both groups.

On comparing the effect of subescharal injection of PVP- Iodine; whether it is superior or not ? Following conclusions were drawn:-

- 1. Subescharal injection of PVP-Iodine causes a slight pain to patients but neither a sedative nor analyseic is required in this procedure.
- 2. Escharolysis was faster in subescharal injected patients and biopsy showed that there was less infection in subescharal plane, as compared to the control group.
- 3. After separation of eschar, healthy granulation surface was seen. Grafts were applied with in a week.

- 4. Graft acceptance in subescharally treated patient was high in comparison to patients not treated with subescharal injection.
- 5. Escharolysis was faster in PVP-Iodine injection treated patient.
- 6. Chance of septicemia is much less in test group.

In brief, the conclusion may be drawn that treating deep burn patients by using subescharal injection of PVP-Iodine is markedly superior as shown by minimal infection rate and markedly reduce escharolysis time. This is due to wide spectrum of action, increase concentration of anti-microbial in subescharal plane and attainment of very few microbial colonies in subescharal plane.



BIBLIOGRAPHY

BIBLIOGRAPHY

- 1. Artz CP, Moncrief JA, Pruitt BA Jr: Burns. A team approach, 1979. W.B. Saunders Co., Philadelphia.
- 2. Artz CP: Management of thermal burns. Mod Med, 26:181, 1960.
- 3. Appel GB and Neu HC: Nephrotoxicity of antimicrobial agents. N Engl J Med, 296: 663-70, 722, 28, 784-87, 1977.
- 4. Baxter GB, Crusin PW Marvin JA: The control of burn wound sepsis by the use of quantitative bacteriologic studies and subescharoclysis with antibiotics. Surg Clin North America, 1973, 53:1504-1518.
- 5. Balogh D, Bauer M, Riccabona G: The influence of povidone treatment on thyroid hormones in severe burns. Journal of Hospital Infection, 1985, 6: 147-153.
- 6. Bell E, Erlich HP, Buttle DJ et al: Living tissue formed in vitro and accepted as skin equivalent tissue of full thickness. Science, 211: 1052, 1981.

- 7. Baxter CR: Homografts and Hetrografts as a biological dressing in the treatment of thermal injury. Presented at the first Annual Congress of the Society of German plastic Surgeons in Munich, Germany, September 28, 1970.
- 8. Bharadwaj R, Joshi BN & Phadke SA: Assessment of burn wound sepsis by swab, full thickness biopsy culture and blood culture a comprehensive study. Burns, 10: 124-126, 1983.
- 9. Bose B: Burn wound dressing with human amniotic membrane. Ann R Coll Surg Engl, 61 (6): 444-7, 1979.
- 10. Brown MRW and Wood SM: Relation between pseudomonas a. protetus V and K. aerogeneses and their sensitivity to poly. B and other antibacterial agents. J pharm Pharmacol, 24: 24: 215-28, 1972.
- 11. Brown MRW and Frayer MP: Post mortem homograft to recude mortality in extensive burrns. JAMA, 165: 1163,1954.
- 12. Bromberg BE, Song IC, Mohn MP: The use of pig skin as a temporary biological dressing. Plast Reconstr Surg, 36: 80, 1965.
- 13. Burke JF, Yannas IV, Quinby WC Jr et al :Artifical skin in treatment of extension burns injury. Ann Surg, 194: 413, 1981.

- 14. Burke JF, Quinby WC Jr, Bondac CC: Primary excision and prompt grafting as routine therapy for treatment of thermal burn in children. Surg Clin North Am, 56: 477-495, 1976.
- 15. Chopra, Handa and Kapur: Indigenous drug of India, 569, 685, 1982.
- 16. Copeland CE; Clinical experience with topical Betadine ointment in the treatment of burn patients. In: Reber H (ed.) "Proceeding of the Second World Congress on Antisepsis", pp. 120-122, H.P. Publishing Co., New York.
- 17. Crinder JH: Skin grafting with graft taken from dead subjects. Med Recurrence N Y, 199, 1881.
- 18. Curtin JA, Petersdost RG and Benett IL: Pseudomonas bacteraemia: Review of ninety one cases. Ann Inter Med, 54: 1077-1107, 1961.
- 19. Dango C: Survival and utilisation of cadaver skin. Plast Reconst Surg, 10: 10, 1952.
- 20. Davidson EC: Tannic acid in the treatment of burn, Surg, Gynaec Obstet, 41: 202, 1925.
- 21. Davis JS: Skin transplantation with a review of 550 cases at the John Hopkins Hospital, J H H report, 15: 307, 1910.
- 22. Demling RH: Improved survival after massive burns. J Trauma, 1983, 23: 179-184.

- 23. Elliott RA, Hoehn JG: Plast Reconst Surg, 52 (4): 401, 1973, Quoted by G.B. Park, 1978.
- 24. Fox CL Jr, Rappole BW, Stenford WT: Control of pseudomonas infection by silver sulphadiazine. Surg Gynae Obster, 128: 1021, 1969.
- 25. Fox CL Zr :Silver sulphadiazine for control of burn wound infection. International Surgery Vol. 60, No. 5, 1975.
- 26. Georgiade NG, Harris WA: Open and closed treatment of burns wound infection.

 International Surgery Vol. 60, No. 5, 1975.
- 27. Gupta RL et al : Role of collagen sheet cover in burn a clinical study. Ind J Surg, 40 : 646-649, 1978.
- 28. Hauben DJ, Yanoi E, Mahler D: On the history of treatment of burns. Burns, 7: 383, 1981.
- 29. Hansen E, Troensegarel: Amniotic grafts in chronic skin ulceratain. Lancet, 1: 859, 1950.
- 30. Ivunova SS: The transplantation of skin from dead body to granulating surface. Ann Surg, 99: 763, 1969.
- 31. James AO, Neel Jr et al : The extended use of skin homografts. Arch Surg, 99: 763, 1969.
- 32. Jullian A Sterling: Use of amniotic membrane to cover surface defects due to flame buns. Ann J Surg, 91: 940, 1956.

- 33. Kock DM: Topical burn therapy comparing povidone iodine ointment or cream plus aserbine and povidone-iodine cream. Journal of hospital Infection, 1985, 6: 127-132.
- 34. Kornberg J, Hume HK, Kofesjiam R et al: Ultra thine silicone polymer membrane: A new synthetic skin substitute. Trans Ann Soc Artif Inter Organ, 18: 39, 1972.
- 35. Krupp S, Barchler M, Bille J: Assessment of burn wound sepsis. Journal of Hospital Infection, 6: 133-137, 1985.
- 36. Law EJ Jr, MacMillan BC: Topical treatment of small burn wounds with povidone-iodine. In:
 Polk HC, Ehrenkrauz NJ (Eds.) "Therapeutic Advances and New Clinical Indications. Medical and Surgical Antisepsis with Betadine Microbicides". The purdue Fredrick Co., Yonkers, N Y, 121: 1972.
- 37. Liedberg NCF, Resis E, Artz CP: Infections in burns III. Septicaemia, a common cause of death, Surg Gynec & Obst, 99: 151-158, 1954.
- 38. Loebi EC Marvin JA, Heak El et al: The method of quantitative burn wound biopsy cultures and it's routine use in case of burn patients. Am J Clin Pathology, 61: 20-24, 1974.

- 39. Mc Manus WF, Manson AD, Pruitt BA. Subeschar antibiotics infusion in treatment of burn wound infection. J trauma, 20: 1021-1023, 1980.
- 40. Mc Manus WF, Goodwin CW Jr, Manson AD Jr et al:
 Burn wound infection. J Trauma, 21: 753-756,
 1981.
- 41. MacMillan BG: Infections following burn injury. Surg Clin North Amer, 60: 185-196, 1980.
- 42. Moncrief JA. A Topical anti-bacterial therapy for burn wounds. Clin Plast Surg, 1: 563, 1974.
- 43. Moncreif AJ, Teplitz C: Changing concepts in burns sepsis. J Trauma, 4: 223-245, 1964.
- 44. Moyer CA, Brantano L, Cravenes DL et al:
 Treatment of large human burns with 5% silver
 nitrate solution. Arch Surg, 90: 812, 1965.
- 45. Moncreif JA, Rivers JA: The problem of infection in burns by resistance micro-organisms. Ann Surg, 7: 295-312, 1958.
- 46. Monofo WM, Tandon SN: A comparitive study of topical antiseptic agents in the treatment of 24 patients with burns. In: Reber H (ed.) "Preceding of the Second World Congress on Antisepsis", pp 163-166; HP Publishing Co., New York.

- 47. Nicholas G Georgide MD and Isidro J Amigo MD Durham NC: Changing concepts in treatment of burn. Southern Medical Journal USA, 56: 337-345, April, 1963.
- 48. Oppenheimer LS: The treatment of burns. N K Med J, 84: 645, 1906.
- 49. Park GB: Burn wound coverings- a review.
 Biometer Med Devices Artif Organ, 6: 1-35,
 1978.
- 50. Pinkerston MC: Amnioplastin for adherent digital flexor tendons. Lancet, 1: 70, 1942.
- 51. Pollock: Quoted by Fresh Water MF and Krizek
 TJ: Skin grafting of burns. A Centennial J
 Trauma, 11: 862, 1971.
- 52. Pratt WB: Chemotherapy of infection. Oxford University Press, New York, 1977 pp 128-175.
- 53. Pruitt BA Jr & Foley AD: The use of the biopsies in burn patient care. Surgery, 73: 887-897, 1973.
- 54. Petter Zellner MD: Reduction of surgery in burn patients treated with betadine ointment.

 Vocational Association Accident Clin Ludwigshafer Oggersherm West Germany.
- S5. Rappoport I, Papine AT and Dietrick W: early use of Xenografts as biological dressing in burn trauma. AMJ Surgery, 120: 144, 1970.

- Robson CM, Schaerf RHM, Krizek TJ: Evaluation of topical povidone-iodine ointment in experimental burn wound sepsis. Plast Reconst Surgery, 54 (3): 328-334, 1974.
- 57. Robert H, Demling MD: Burn, The new England Journal of Medicine, Vol 313; 22: 1389-1396;1985.
- 58. Sabella N: Use of foetal membrane in skin grafting. Med Recurrence N Y, 83: 478, 1913.
- 59. Salisbury RE, Wilmoro DM, Silverstein P et al: Biological dressing for skin grafting donor site. Arch Surg, 106: 705, 1973.
- 60. Schwartz, Shires and Spencer: Principles of Surgery. Ved. Page 294, 295, 1998.
- 61. Shanker ML: A clinical study on the use of collagen sheet in the management of burn wounds. Thesis for M S Examination Surgery.

 Bangalore University, Bangalore, 1975.
- Sharma et al : Evaluation of use of preserved skin homograft in patients of burns. Thesis for M S Examination Surgery, Agra University, Agra, 1978.
- 63. Shede M VII Veseins Chronik: Arctlicher Verein Zu Hamburg, Sitzung Von 25. Jr 1881 Dtrch Med Wonchenchr, 7: 352, 1881.

- 64. Sinha Rajeev et al : A new approach to the management of burn injury Using PVP+ Neosporin.

 Int Surg, 73: 126-129, 1988.
- 65. Sinha RM, Verma PK and Madam P: Collagen sheet as biological dressing in burns. Ind J Plast Surg 5(2)1972.
- 66. Silverstein P, Curreri PW and Master AM:
 Evaluation of fresh viable porcine, cutaneous
 xenograft as a temporary burn wound cover.
 Annual Progress Report, US Army Institute of
 Surgical Research, 1970-71.
- 67. Trelford JD, Hanson FW and Anderson DG:
 Amniotic membrane as a living surgical dressing
 in human patients. Onchology, 28: 358, 1973.
- 68. Tavis MJ, Thornton J, Danet R et al: Current status of skin substitute. Surg Clin North Am, 58: 1233, 1978.
- 69. Weinsten L: Bacitracin and Polymyxin B, In:
 Goodman LS and Gillman A(eds.) The
 pharmacological basis of therapeutics, IV ed.,
 N. York, MacMillan and Co., 1970. P 1292 and
 1287.
- 70. William WM, Bruce F: Topical therapy fo rburns. Surg Clin North Amer, 67 (1): 133-145, 1987.

- 71. William W Monofo, Som N Tondon: Cerium nitrate-A new topical antiseptic for extensive burns. Surg, 80 (5), 1975.
- 72. Zamora LJ: Povidone-Iodine and wound infection, Surgery, 1984; Jan: 121-122.
- 73. Zellner PR, Bugyi S: Povidone-Iodine is the treatment of burn patients. Journal of Hospital Infection, 6:139-146, 1985.